

An Outbreak of Illness Among Aerospace Workers

PATRICIA J. SPARKS, MD, MPH; GREGORY E. SIMON, MD; WAYNE J. KATON, MD; LEONARD C. ALTMAN, MD;
GARRISON H. AYARS, MD; and RICK L. JOHNSON, MD, *Seattle*

A multispecialty panel of physicians evaluated a case series of 53 composite-materials workers in a large aircraft manufacturing facility who filed workers' compensation claims for illness labeled by the media as the "aerospace syndrome." Possible skin and respiratory tract exposures included formaldehyde, phenol, particulates, epoxy resins, and trace organic solvents, but measured concentrations were well below all regulatory and consensus standards. Most workers had histories of transient skin or respiratory tract irritation consistent with the known potential toxicity of these materials. None of the workers tested had immunoglobulin IgG or IgE antibodies to human serum albumin complexed with formaldehyde. A majority (74%) met DSM-III-R [*Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised] criteria for major depression, panic disorder, or both. Most of these psychiatric disorders were of a recent onset, correlating in time with the use of phenol- and formaldehyde-impregnated composite material. Psychosocial factors were thought to have played a major role in the high prevalence of illness in this group and should be evaluated directly in well-controlled epidemiologic studies of similar crisis-building situations in the future.

(Sparks PJ, Simon GE, Katon WJ, et al: An outbreak of illness among aerospace workers. *West J Med* 1990 Jul; 153:28-33)

By mid-1988, more than half of approximately 200 employees working with composite plastic materials in one building of a large aircraft manufacturing company reported multiple symptoms including dizziness, nausea, headaches, fatigue, shortness of breath, palpitations, and cognitive impairment. The composite materials were composed of fiberglass, graphite, and other synthetic fibers. These fibers were impregnated with epoxies or phenol-formaldehyde resins and cured with heat to form rigid aircraft parts. For most workers, the symptoms were reported to begin within one week to six months after the introduction of fiberglass cloth impregnated with phenol-formaldehyde resin (phenolic material) in mid-1987 to comply with Federal Aviation Administration regulations regarding fire retardation.

Most of the workers reporting symptoms had been referred by their fellow workers and the local union to an allergist in the community. Some workers had been referred by the allergist to a psychiatrist who diagnosed permanent organic brain damage said to be "typical" of toxic chemical exposure. The allergist announced to the media the presence of a new disease, the "aerospace syndrome." Workers understood from these physicians that they had antibodies to formaldehyde, immune system dysfunction, and brain damage. This prompted several months of local and national media coverage, intense union-management deliberations, and a United States Senate subcommittee hearing on the issue.

After industrial hygiene evaluation, the cause of the new "aerospace syndrome" remained unclear. Despite industrial hygiene data showing workplace chemical exposures well below those typically considered risks to health, an increasing number of workers became ill and disabled. The manufacturer and the workers' compensation administrator enlisted one of us (P.J.S.) to conduct an independent medical evaluation of all of the workers filing compensation

claims. A multidisciplinary panel was convened and included specialists in occupational medicine-clinical toxicology, allergy-immunology, and psychiatry with consultants in neurology, dermatology, and pulmonary medicine. The following is a case-series presentation of the clinical findings.

Patients and Methods

Industrial Process and Exposure Monitoring

Those involved in the outbreak worked primarily with fiberglass impregnated with phenol-formaldehyde resin, although epoxy resins had also been used in the same area for several years. The process, called "lay-up," involved hand molding and oven curing of the composite material. Some parts required reworking with saws and routers and filling in defects with epoxy or phenolic compounds. Small amounts of organic solvents were used for cleaning the parts and molds. Suspected chemicals included phenol, formaldehyde, epoxy resins, and trace amounts of styrene, antimony trioxide, methylene chloride, acetone, C9 through C12 alkanes, C9 through C12 aromatics, salicylaldehyde, and methanol. Disposable plastic laboratory coats, sleeves, and gloves were available (but optional) until the spring of 1988 when the use of gloves became mandatory. Half-mask respirators with organic vapor-acid gas cartridges were also made available at that time.

During 1987 and again in the spring of 1988, extensive personal air monitoring for phenol, formaldehyde, total particulates, and antimony trioxide was obtained by the company using industrial hygiene methods published by the National Institute of Occupational Safety and Health (NIOSH).¹ During the summer of 1988, similar air monitoring, which also included styrene, acetone, and methylene chloride, was carried out by the Washington State Division of Occupational Safety and Health and NIOSH using the same methods. Thermatogravimetric and gas chromatography-mass spectrometry were also used to analyze

From the Occupational Health Services, Providence Medical Center (Dr Sparks), and the Department of Psychiatry (Drs Simon and Katon) and the Departments of Medicine (Drs Altman, Ayars, and Johnson) and Environmental Health (Dr Altman), Division of Allergy and Infectious Diseases, University of Washington School of Medicine, Seattle. Dr Simon is a Robert Wood Johnson Clinical Scholar in Psychiatry.

Reprint requests to Patricia J. Sparks, MD, MPH, Director, Occupational Health Services, Providence Medical Center, 500 17th, C-34008, Seattle, WA 98124.

ABBREVIATIONS USED IN TEXT

AIDS = acquired immunodeficiency syndrome
 DIS = Diagnostic Interview Schedule
 ELISA = enzyme-linked immunosorbent assay
 GC-MS = gas chromatographic-mass spectrophotometric
 HSA = human serum albumin
 NIOSH = National Institute of Occupational Safety and Health

graphic-mass spectrophotometric (GC-MS) analysis² of the phenolic material was also done by the University of Washington Department of Environmental Health, the aerospace company, and NIOSH.

Worker Selection

In all, 60 workers filed compensation claims during 1988 for health complaints possibly related to work with the phenolic material. Seven workers failed to report for examination. Of the no-shows, six said that their symptoms, involving multiple organ systems, had completely resolved. Evaluations of a total of 53 workers with exposure to phenol-formaldehyde composite materials are included in this report.

Clinical Evaluation

Each worker was initially seen by an occupational medicine specialist-internist. A detailed occupational and medical history was obtained, and a physical examination, including a neurologic examination, was done. Most of the workers were also evaluated by an allergist-immunologist. Current and past medical records, before occupational exposure, were reviewed. Blood specimens were drawn for liver, kidney, and thyroid function studies, electrolytes, and complete blood count with differential. A urinalysis and screening spirometry were obtained.

Immunologic Evaluation

Serologic assays for formaldehyde complexed with human serum albumin (HSA-formaldehyde) were analyzed at Northwestern University Division of Allergy/Immunology by enzyme-linked immunosorbent assay (ELISA) according to previously published methods.³ This test, done on specimens from the first 33 workers filing compensation claims, measures titers of immunoglobulin IgG and IgE antibodies to HSA-formaldehyde and HSA alone in the serum of the subjects. The test was standardized using serum from three known negative controls and a positive control. The specimens were analyzed by two senior technicians using coded specimens without knowledge of clinical information. Any serum producing an optical density greater than twice that of the control serum was retested twice by each technician.⁴

Neuropsychiatric Evaluation

Each subject completed several self-report questionnaires. These included the SCL-90-R,⁵ the Whiteley Index,⁶ and a questionnaire devised by one of us (G.E.S.) to assess symptoms on exposure to a variety of chemicals. The SCL-90-R is a 90-item checklist assessing the presence of somatic and psychological symptoms on a 0 to 4 severity index. Raw scores were converted to standardized (T-score) norms for nonpatient populations.⁵ The Whiteley Index (a version of Pilowsky's Illness Behavior Questionnaire) is a 14-item self-report scale assessing hypochondriacal beliefs and behavior. It has been found to discriminate between hypochondriacal patients and medical controls. Last, the patients completed a questionnaire assessing the presence of symptoms of "multiple chemical sensitivity." This was

defined as the reporting of multiple somatic symptoms on exposure to low levels of a variety of chemically unrelated substances (such as automobile exhaust, perfumes, new carpet odor, and newsprint), in both the occupational and nonoccupational settings, that were not associated with objective findings of organ system impairment or physiologic dysfunction.⁷

All subjects also underwent a structured psychiatric evaluation using the National Institute of Mental Health's Diagnostic Interview Schedule (DIS).⁸ The DIS is a highly structured interview that investigates the presence of symptoms of major psychiatric illnesses and assigns current (within the past six months) and past diagnoses according to criteria^{8,9} of the *Diagnostic and Statistical Manual of Mental Disorders*, third edition, revised. The DIS was used instead of a free-flowing psychiatric interview to allow a more objective and standardized evaluation of the presence of psychiatric illness. The interview protocol systematically evaluates each reported symptom to determine its relation to medical illness, drug or alcohol use, or psychiatric illness. One section of the interview specifically assesses how often respondents have sought care for medically unexplained physical symptoms. The DIS interview includes Folstein's Mini-Mental State examination,¹⁰ a brief test of cognitive ability. This instrument has excellent reliability and validity and has been found to correlate well with more extensive neuropsychological testing.¹¹ Five patients were interviewed by two psychiatrists to ensure uniformity of use of the DIS. Past psychiatric diagnoses were determined by the reporting of past psychiatric symptoms on the DIS and a review of past medical records.

After these examinations, workers with dermatologic signs were evaluated by a dermatologist. Those with Mini-Mental State examination scores of 25 or less or with complaints of cognitive dysfunction were subsequently referred for evaluation with a standard battery of neuropsychological tests.¹¹ These included measures of attention and concentration, executive control and problem solving, language, visuospatial orientation, memory, sensory perception, motor function, cognition, emotion, personality, and motivation. Because depression and anxiety may cause mild abnormalities of cognitive function on neuropsychological testing,¹¹ all subjects underwent a psychiatric evaluation before such testing. Workers with a subjective sensory loss were evaluated by a neurologist and underwent nuclear magnetic imaging of the head, nerve conduction studies, or both. Those with symptoms of airway reactivity and normal spirometric values underwent methacholine challenge testing.

Results*Air Monitoring Data*

Thermogravimetric GC-MS analysis of the phenolic material failed to reveal the presence of significant quantities of unsuspected materials. Airborne levels of phenol, formaldehyde, antimony trioxide, total particulates, styrene, and other trace organic compounds measured by the company, the state, and NIOSH were well below federal permissible exposure limits (Table 1). Those filing compensation claims did not have significantly different exposures from those in the same work area who did not.

Clinical Information

Of the 53 workers, 44 (83%) were women and 27 (51%) were currently cigarette smokers. Smoking did not affect the number of reported symptoms. There was no difference in the range of symptoms reported by the 49 workers who

consulted the local allergist compared with the 4 workers who did not. Symptoms did not vary significantly by job category, duration of exposures, or location within the composite production area. The exposure concentrations were so uniformly low, it was not possible to distribute workers into meaningful exposure groups so that a dose-response relationship of symptoms and exposures could be assessed.

The summary of the clinical findings appears in Tables 2 and 3. Sensory or upper respiratory tract irritation (or both) was seen in 39 (74%) workers. Sensory irritation was defined as headache or nausea associated with the odor of the phenolic material. Nineteen (36%) also reported dermatitis. A history of skin rash was associated with an initial lack of glove use. Most of the workers with dermatitis also had symptoms of upper respiratory tract or sensory irritation.

Fifteen workers (28%) described symptoms consistent with the "multiple chemical sensitivity syndrome."⁷ Four of the workers (8%) described sensory loss in the extremities that was not confirmed by objective neurologic signs or diagnostic testing.

In general, the physical examinations of these workers revealed few objective findings. Five of seven workers with findings of upper respiratory tract irritation had histories of atopic disease. Eight workers had evidence on spirometry, or methacholine challenge, of reactive airways disease. In five of these workers, this was explained by either previously symptomatic atopy or asthma. Results of the other laboratory tests (complete blood count, liver and kidney functions, electrolytes) were generally unremarkable.

Immunologic Evaluation

The first 33 subjects had analyses of serum by ELISA to measure serum levels of IgE and IgG antibodies to HSA-formaldehyde. There were no differences between workers' results and controls. For this reason and because of the high cost of this assay, the test was not done on the rest of the group.

Neuropsychiatric Evaluation

The most striking finding of the psychiatric evaluations was the high prevalence of anxiety and depressive disorders (Table 4). Only 14 subjects had no diagnosis of depression or panic disorder associated with their current symptoms. The high prevalence of current psychiatric diagnoses correlated in time with work with phenol-formaldehyde composites and was not well explained by preexisting psychiatric disease (Table 4). Scores on the SCL-90-R also showed a high level of current psychiatric symptoms. Most subjects had scores in all subscales except paranoia that were greater than two standard deviations above the mean for the general population. All scales showed similar degrees of elevation. For most, current psychiatric symptoms had not lessened substantially with removal from work with phenol-formaldehyde composites at the time of this evaluation.

A subset of subjects, most with past symptoms of psychiatric disorders, appeared to have a preexisting tendency toward somatization (Table 4). Three qualified for the diagnosis of somatization disorder⁹ by the DIS interview and a review of medical records. Using an abridged definition of somatization proposed by Escobar and co-workers (four medically unexplained symptoms for a man and six for a woman),¹² 14 subjects had a previous tendency to present with medically unexplained physical symptoms thought to be based on psychological distress and had Whiteley Index scores typical of hypochondriacal persons. Most of these were the same subjects who met our definition of having the "multiple chemical sensitivity syndrome,"⁷ whose charac-

teristics are described further in another publication.¹³ In the abridged definition, somatization (somatization trait) has been shown to be associated with an increased and preferential use of medical versus mental health services when psychiatric illness is present. For the entire group, the mean score on the Whiteley Index was 5.0, lying between

TABLE 1.—Summary of the Air Monitoring Data in 53 Aerospace Workers With Workplace Exposures

Substance	Measured Values	Permissible Exposure Limits*
Phenol, ppm.	0.001-0.180	5
Formaldehyde, ppm.	0.003-0.073	1
Antimony trioxide, mg/m ³	BDL	0.5
Total particulates, mg/m ³	0.48-1.1	10
Styrene, ppm.	BDL-1.2	100
Methylene chloride, ppm.	BDL-0.1	100
Acetone, ppm.	BDL-7.6	1,000

BDL=below detection level for method used, ppm=parts per million

*Federal Occupational Health and Safety Administration Standards.

TABLE 2.—Summary of Subjective Complaints in 53 Aerospace Workers With Workplace Chemical Exposures

Symptom	Subjects,	
	Number	%
History of contact dermatitis.	19	36
Episode of hives.	3	6
Sensory irritation.	39	74
Eye and upper respiratory tract irritation.	30	57
Lower respiratory tract irritation.	19	36
Gastrointestinal symptoms.	8	15
"Multiple chemical sensitivity"	17	32
Numbness of extremities.	4	8
Multiple somatic complaints.	16	30
Difficulty with memory and concentration.	22	42

TABLE 3.—Summary of Objective Findings in 53 Aerospace Workers With Workplace Chemical Exposures

Sign	Subjects,	
	Number	%
Contact dermatitis.	2	4
Upper respiratory tract inflammation.	7	13
Airway reactivity on spirometry or methacholine challenge.	8	15
Elevated serum thyroxine.	4	8
Hematuria.	3	6

TABLE 4.—Summary of Neuropsychiatric Findings in 53 Aerospace Workers With Workplace Chemical Exposures

Finding	Subjects,	
	Number	%
Current depression.	32	60
Past depression.	12	23
Current panic disorder.	14	26
Current panic attacks.	3	6
Past panic disorder.	2	4
Somatization disorder.	3	6
Somatization trait.	14	26
Abnormal findings on Mini-Mental State* examination.	4	8

*The Mini-Mental State examination is part of the National Institute of Mental Health's Diagnostic Interview Schedule (Robins et al⁸).

scores typical of hypochondriacal patients and normal non-patient controls.⁶

Four of the workers had abnormal findings on the Mini-Mental State examinations. For most, the deficit consisted of the inability to subtract 7's from 100. Subsequently, 25 of the workers with abnormal Mini-Mental State examinations, neuropsychological symptoms (such as reduced memory and difficulty with concentration), or both, were referred for detailed neuropsychological testing. None were reported to have significant cognitive deficits of recent onset or correlated with exposure to phenol-formaldehyde composites.

Discussion

More than half of subjects had histories of local symptoms consistent with contact dermatitis, mucous membrane irritation, or both. A few showed physical signs of such irritation at the time of this evaluation. These symptoms and signs were consistent with the known toxicities of the potential chemical exposures present at the work site.¹⁴⁻¹⁹ Most of these local irritant symptoms resolved rapidly without long-term sequelae following removal from exposure.

In contrast, many of the workers had chronic and systemic health complaints that were not consistent with the known toxicity of the materials to which they likely had exposure. For example, it is known that skin exposure to formaldehyde can produce both irritant and allergic contact dermatitis and probably urticaria.¹⁴ Formaldehyde has also been causally related to upper and lower respiratory tract irritation, and rare cases of asthma have been documented in persons with ongoing exposure.¹⁴⁻¹⁶ There are, however, no adequately controlled studies to support the contention that subacute or chronic formaldehyde exposure can produce systemic symptoms such as fatigue, a loss of memory, difficulty in concentrating, or depression.¹⁴⁻¹⁹ There is no evidence that low-level formaldehyde exposure causes organic brain dysfunction or abnormal neuropsychological test results.¹⁴⁻²⁰ The rapid metabolism of formaldehyde makes systemic effects unlikely.²¹ Moreover, smokers have exposure to relatively high levels of formaldehyde,²² but cognitive dysfunction has not been reported as more prevalent in this group.

Other chemicals to which these workers could have been exposed, such as phenol, styrene, acetone, or antimony trioxide, may be associated with systemic symptoms that follow a dose-response relationship.¹⁹ Symptoms associated with acute exposure, such as headache, respiratory tract irritation, or nausea, are usually temporary and abate with removal from exposure. Very high exposure over several years may be associated with irreversible impairment of the nervous system, liver, or kidneys, which can be documented with objective tests.^{19,23} Such effects have been observed with much higher concentrations and longer durations of exposure than has occurred in this setting.

The potential exposures in this workplace do not include exotic chemicals with which occupational medicine has little experience. Low-level exposure to these substances, individually and as chemical mixtures, occurs every day in hundreds of industries without incident.

The diagnosis of "mass hysteria" is made with specific criteria—which include a rapid onset, verbal or visual transmission of illness, and a predominance of symptoms of hyperventilation—that do not entirely explain the timing or the range of symptoms and signs in this group of workers.²⁴ On the other hand, this outbreak of illness has much in common with "crisis building" situations described by

Baker²⁴ in which the onset of illness is slower with a wider range of symptoms reported.

A thorough medical evaluation failed to reveal objective abnormalities that might explain these workers' disabling symptoms. The immunologic evaluation was similarly unrevealing. Most of the subjects reported previous unusual immunologic tests, ordered by a local allergist who had cared for many of the workers affected by this outbreak and carried out by a commercial laboratory. Results of these tests indicated low titers (1:4 to 1:32) of IgG or IgM antibodies to formaldehyde, trimellitic anhydride, or isocyanates complexed with HSA, as well as "elevations" in the number or proportion of TA-1 receptor-positive lymphocytes. Various "positive" autoantibodies were also reported to have been found. Most workers believed that such results indicated that their exposures to "chemicals" were "high" and that their symptoms resulted from "chemical poisoning" or "allergy to chemicals." Some described concern about "chemical AIDS [acquired immunodeficiency syndrome]."

A careful review of the literature raises many questions about the pathophysiologic significance of this battery of immunologic studies. The reported presence of antibodies to HSA-formaldehyde was not replicated by this study's controlled evaluation, which suggests that either the antibodies had disappeared (some workers had been removed from work with phenol-formaldehyde composites), antibodies were never present at all, or there were significant differences in methodology between the commercial and university laboratories. Patterson and associates have said that there is insufficient evidence to support a relationship between gaseous exposure to formaldehyde and the presence of antibodies to HSA-formaldehyde and systemic hypersensitivity or illness.²⁵ In addition, low titers of autoantibodies frequently may be found in healthy persons.²⁶

The use of such an extensive battery of laboratory tests to support an immunologic basis for illness in these workers is unsubstantiated.²⁷ For many of the tests used, there are little data to indicate what a normal result should be for healthy persons. A wide variation in the types of "abnormal findings" among subjects with similar clinical syndromes suggests that such findings may represent chance occurrence. Immunologic data can only be accepted as useful in the diagnosis of illness related to chemical exposure when well-designed epidemiologic studies reveal consistent correlations of such measures with specific exposures and disease states. Existing reports use inadequate controls and fail to consider important factors of case selection, smoking, other chemical exposures, and the presence of other illness such as atopy.²⁸⁻³⁰ Finally, these test batteries are costly.

The most striking finding from this group evaluation was the 74% prevalence of major depression, panic disorder, or both, in these workers. A majority of workers had psychiatric illness develop that was correlated in time with exposure to the phenolic material and a confluence of psychosocial factors. If only symptoms predating work with the phenolic material were considered, the prevalence rates of preexisting psychiatric diseases were 4% for panic disorder and 23% for major depression. These prevalence figures are considerably higher than those found in the general population (3% to 4% for depression),³¹ but they are similar to rates reported for patients visiting general medical clinics.³²⁻³⁴ Thus, this group of workers displayed a high prevalence of anxiety and depression not explained by pre-existing psychiatric illness.

Psychiatric diagnoses in this group probably explain many of the somatic symptoms such as fatigue, difficulty

with concentration and memory, sleep disturbances, dizziness, and palpitations. Patients with major depression and panic disorder have been found to use more nonpsychiatric medical care³⁵⁻³⁷ and to complain of more symptoms³⁷⁻³⁹ than do nondepressed controls. The tendency for affective illness to lead to increased somatic symptoms, a poorer perception of one's health, and an increased perception of disability and impairment in a person's vocational and social roles has been described previously.⁴⁰ Symptoms of depression or panic in many of these workers may have been exacerbated by the mistaken impression that they suffered from "brain damage" or were "poisoned," their immune system was damaged, or that they had "chemical AIDS" because of their occupational exposure to "chemicals."

The high incidence of new cases of panic disorder suggests that there may be significant interaction between the sensory and irritant stimulation of the mucous membranes of the upper respiratory tract and the autonomic or central nervous system reaction to such stimulation. Numerous subjects described episodes of dizziness, palpitations, dyspnea, and faintness occurring at times of exposure to volatile materials associated with a pungent odor. In many subjects experiencing such attacks, clinical syndromes then developed that were indistinguishable from typical panic disorder.

Previous case reports describe the precipitation of panic disorder by exposure to solvents or other respiratory irritants with a noxious odor.^{41,42} Such reactions are difficult to explain by a direct toxic effect of the specific chemicals on the central nervous system. Some of the substances precipitating these reactions, such as chlorine or formaldehyde,^{19,21} are not systemically absorbed. Instead, irritant symptoms coupled with a fear of toxicity may produce a state of autonomic arousal leading to a panic attack. Such a chain of events can be postulated to lead to a conditioned response in which subsequent exposures could produce similar panic attacks with their associated physical symptoms.⁴³ This mechanism has been described by Shusterman and colleagues as "behavioral sensitization to odorants."⁴⁴

Our findings suggest that psychiatric morbidity may explain much of the illness and disability experienced by this group of workers. There is accumulating evidence that long-term exposure to mixed organic solvents can cause organic brain syndromes with anxiety and depressive symptoms as prominent components.^{23,45-49} In these studies, however, workers with solvent neurotoxicity had exposure to far higher levels of solvents for much longer durations than the subjects of this report. Although we cannot absolutely exclude that this outbreak of symptoms of anxiety and depression could have resulted from unknown neurotoxic effects of very low levels of mixed chemical exposures, this possibility seems unlikely.

This study is most limited by the fact that all patients were seen during a workers' compensation evaluation process and that this circumstance precluded the evaluation of a control group. Symptom reporting in a subset of this group of workers with exposure to phenol-formaldehyde composite materials was compared with that of a similar group of workers in another facility within the same company not working with this material.⁵⁰ This comparison showed that the employees working with phenol-formaldehyde had a higher proportion of female workers, had worked for the company a shorter time, and had a higher prevalence of self-reported respiratory tract symptoms and hand rashes than the reference group. The prevalence of psychiatric symptoms was not addressed, however.

Some workers clearly considered psychiatric evaluation

as an attempt to prove the psychological origin of their symptoms and thus deny them compensation. Such a bias would likely have resulted in a falsely lowered prevalence of psychiatric illness and psychological morbidity. In contrast, the finding of high levels of psychiatric morbidity in this population might have resulted from ill workers with psychological symptoms being more likely to seek workers' compensation. If such selection occurred, psychiatric illness would be overrepresented among this group as compared with other workers affected by this same outbreak who did not file claims. Even if none of the other affected workers had psychiatric illness, however, the prevalence of such illness is higher than expected.³¹

Psychosocial factors in the workplace and the community are likely to have been major contributors to this outbreak of illness. A recent study of a similar outbreak reported that work intensity and mental strain were the most important variables predicting severity of illness.⁵¹ Many of our subjects described such factors as increased production pressure, tense labor-management relations, worker perception of inadequate management attention to safety, and reinforcement of fear by co-workers, the media, and medical providers. Many workers were alarmed by the perception that their immune system was damaged and that they were poisoned or seriously ill. Psychosocial factors should be evaluated directly in well-controlled epidemiologic studies of similar crisis-building situations in the future.

Whether these psychiatric symptoms were caused by sensory or respiratory tract irritation from low-level chemical exposure, or primarily psychosocial factors and the effects of the fear of chemicals, they have clearly caused much distress and functional disability. There are specific and effective treatments that can relieve the psychiatric and physical distress generated by these illnesses.^{34,35,37} Unfortunately, the label "aerospace syndrome," and the avoidance of all chemical exposures and the workplace recommended by some health care practitioners, may only perpetuate illness and reinforce disability.⁵²

REFERENCES

1. Manual of Analytic Methods, publication No. 84-100. Washington, DC, US Dept of Health and Human Services, National Institute of Occupational Safety and Health, 1984
2. Kalman DA: Survey analysis of volatile organics released from plastics under thermal stress. *Am Ind Hyg Assoc J* 1986; 47:270-275
3. Patterson R, Harris KE, Grammer LC: Canine antibodies against formaldehyde-human serum albumin conjugates: Induction, measurement, and specificity. *J Lab Clin Med* 1985; 106:93-100
4. Grammer LC, Harris KE, Shaughnessy DS, et al: Clinical and immunologic evaluation of 37 workers exposed to gaseous formaldehyde. *J Allergy Clin Immunol*, in press
5. Derogatis LR: The SCL-90 Scoring Manual I: Scoring, Administration, and Procedures for the SCL-90. Baltimore, Johns Hopkins University School of Medicine, Clinical Psychometrics Unit, 1977
6. Pilowsky I: Dimensions of hypochondriasis. *Br J Psychiatry* 1967; 113:89-93
7. Cullen MR: The worker with multiple chemical sensitivities: An overview. *State Art Rev Occup Med* 1987; 2:655-661
8. Robins LN, Helzer JE, Croughan J, et al: National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Arch Gen Psychiatry* 1981; 38:381-389
9. Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, revised. Washington, DC, American Psychiatric Association, 1987
10. Folstein MF, Folstein SE, McHugh PR: 'Mini-mental state'—A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975; 12:189-198
11. Hartman DE: *Neuropsychological Toxicology*. New York, Pergamon Press, 1988
12. Escobar JI, Burnam A, Karno M, et al: Somatization in the community. *Arch Gen Psychiatry* 1987; 44:713-718
13. Simon GE, Katon WJ, Sparks PJ: Allergic to life: Psychologic factors in environmental illness. *Am J Psychiatry*, in press
14. Bardana EJ, Montanaro A: The formaldehyde fiasco: A review of the scientific data. *Immunol Allergy Pract* 1987; 9:11-24
15. Feinman SE: *Formaldehyde Sensitivity and Toxicity*. Boca Raton, Fla, CRC Press, 1988

16. National Center for Toxicological Research: Consensus workshop report on formaldehyde, Little Rock, AK. *Environ Health Perspect* 1984; 58:323-381
17. Deichman WB, Keplinger ML: Phenols and phenolic compounds, *In* Clayton DG, Clayton FE (Eds): *Patty's Industrial Hygiene and Toxicology*. New York, John Wiley & Sons, 1981, pp 2567-2627
18. Barrow CS, Steinhagen WH, Cheng JCF: Formaldehyde sensory irritation, *In* Gibson JE (Ed): *Formaldehyde Toxicity*. New York, Hemisphere, 1983
19. Clayton DG, Clayton FE (Eds): *Patty's Industrial Hygiene and Toxicology*. New York, John Wiley & Sons, 1981
20. Cripe LI, Dodrill CB: Neuropsychological test performances with chronic low-level formaldehyde exposure. *Clin Neuropsychol* 1988; 2:41-48
21. Casanova-Schmitz M, Starr TB, Heck HD: Differentiation between metabolic incorporation and covalent binding in the labeling of macromolecules in the rat nasal mucosa and bone marrow by inhaled [^{14}C]- and [^3H]formaldehyde. *Toxicol Appl Pharmacol* 1984; 76:26-44
22. Ayer HE, Yeager DE: Irritants in cigarette smoke plumes. *Am J Public Health* 1982; 72:1285-1285
23. Baker EL, Fine LF: Solvent neurotoxicity: The current evidence. *J Occup Med* 1986; 28:126-129
24. Baker DB: Social and organizational factors in office building-associated illness, *In* Cone JE, Hodgson MF (Eds): *Problem Buildings: Building Associated Illness and the Sick Building Syndrome*. State Art Rev Occup Med 1989; 1:607-624
25. Patterson R, Dykewicz MS, Grammer LC, et al: Formaldehyde reactions and the burden of proof (Editorial). *J Allergy Clin Immunol* 1987; 79:705-706
26. Tomer Y, Shoenfeld Y: The significance of natural autoantibodies. *Immunol Invest* 1988; 17:389-424
27. Terr AI: 'Multiple chemical sensitivities': Immunologic critique of clinical ecology theories and practice, *In* Cullen MR (Ed): *Workers With Multiple Chemical Sensitivities*. State Art Rev Occup Med 1987; 2:683-694
28. Broughton A, Thrasher JD: Antibodies and altered cell mediated immunity in formaldehyde exposed humans. *Common Toxicol* 1988; 2:155-174
29. Thrasher JD, Broughton A, Micevich P: Antibodies and immune profiles of individuals occupationally exposed to formaldehyde: Six case reports. *Am J Ind Med* 1988; 14:479-488
30. Broughton A, Thrasher JD, Gard Z: Immunological evaluation of four arc welders exposed to fumes from ignited polyurethane (isocyanate) foam: Antibodies and immune profiles. *Am J Ind Med* 1988; 13:463-472
31. Robins LN, Helzer JE, Weissman MM, et al: Lifetime prevalence of specific psychiatric disorders in three sites. *Arch Gen Psychiatry* 1984; 41:949-958
32. Kessler LG, Burns BJ, Shapiro S, et al: Psychiatric diagnoses of medical service users: Evidence from the epidemiologic catchment area program. *Am J Public Health* 1987; 77:18-24
33. Kessler LG, Cleary PD, Burke JD: Psychiatric disorders in primary care. *Arch Gen Psychiatry* 1985; 42:583-587
34. Barrett JE, Barrett JA, Oxman TE, et al: The prevalence of psychiatric disorders in a primary care practice. *Arch Gen Psychiatry* 1988; 45:1100-1106
35. Regier DA, Hirschfeld RMA, Goodwin FK, et al: The NIMH Depression Awareness, Recognition, and Treatment Program: Structure, aims and scientific basis. *Am J Psychiatry* 1988; 145:1351-1357
36. Thompson JW, Burns BJ, Bartko J, et al: The use of ambulatory services by persons with and without phobia. *Med Care* 1988; 26:183-198
37. Katon W: Panic disorders and somatization: A review of 55 cases. *Am J Med* 1984; 77:101-106
38. Mathew RJ, Weinman ML, Mirabi M: Physical symptoms of depression. *Br J Psychiatry* 1981; 139:293-296
39. Waxman HM, McCready G, Weinrot RM, et al: A comparison of somatic complaints among depressed and nondepressed older persons. *Gerontologist* 1989; 25:501-507
40. Wells K, Golding JM, Burnham MA: Psychiatric disorders and limitations of functioning in a sample of the Los Angeles general population. *Am J Psychiatry* 1988; 145:712-717
41. Bolla-Wilson K, Wilson RJ, Bleecker ML: Conditioning of physical symptoms after neurotoxic exposure. *J Occup Med* 1988; 30:684-686
42. Dager SR, Holland JP, Cowley DS, et al: Panic disorder precipitated by exposure to organic solvents in the work place. *Am J Psychiatry* 1987; 144:1056-1058
43. Schottenfeld RS, Cullen MR: Recognition of occupational induced post-traumatic stress disorders. *J Occup Med* 1986; 24:365-369
44. Shusterman D, Balmes J, Cone J: Behavioral sensitization to irritants/odorants after acute overexposures. *J Occup Med* 1988; 30:565-567
45. Cherry N, Hutchins H, Pace T, et al: Neurobehavioral effects of repeated occupational exposure to toluene and paint solvents. *Br J Ind Med* 1985; 42:291-300
46. Hane M, Axelsson O, Blume J, et al: Psychological function changes among house painters. *Scand J Work Environ Health* 1977; 3:91-99
47. Hänninen H, Eskelinen L, Husman K, et al: Behavioral effects of long-term exposure to a mixture of organic solvents. *Scand J Work Environ Health* 1976; 2:240-255
48. Lindström K, Riihimäki H, Hänninen K: Occupational solvent exposure and neuropsychiatric disorders. *Scand J Work Environ Health* 1984; 10(suppl):321-323
49. Struwe G, Wennberg A: Psychiatric and neurological symptoms in workers occupationally exposed to organic solvents—Results of a differential epidemiological study. *Acta Psychiatr Scand (Suppl)* 1983; 67:68-80
50. Health Hazard Evaluation Report, publication No. HETA 88-294-1974. Washington, DC, US Dept of Health and Human Services, National Institute of Occupational Safety and Health, 1989
51. Hall EM, Johnson JV: A case study of stress and mass psychogenic illness in industrial workers. *J Occup Med* 1989; 31:243-250
52. American College of Physicians: Position Paper: Clinical ecology. *Ann Intern Med* 1989; 111:168-177